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FILE 'HOME' ENTERED AT 16:18:34 ON 21 JAN 2005

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=> s hypercalciuria
L1 7830 HYPERCALCIURIA

=> s l1 and risk
L2 1061 L1 AND RISK

=> s hypercalciuria and (increase risk or predisposition or risk factor or disease risk?)

6 FILES SEARCHED...
L3 342 HYPERCALCIURIA AND (INCREASE RISK OR PREDISPOSITION OR RISK FACTOR OR DISEASE RISK?)

=> d l2 and increase risk
'AND' IS NOT A VALID FORMAT
'INCREASE' IS NOT A VALID FORMAT
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=> s l2 and increase risk
L4 1 L2 AND INCREASE RISK

=> d l4 ti abs ibib tot

L4 ANSWER 1 OF 1 USPATFULL on STN
TI Rapid identification of conditions, compounds, or compositions that inhibit, prevent, induce, modify, or reverse transitions of physical state
AB The invention concerns arrays comprising hundreds, thousands, to hundreds of thousands of samples and methods for screening thereof. These methods are useful to optimize, select, and discover compounds, compositions, or conditions that prevent, inhibit, induce, modify, or reverse physical-state transitions, particularly in-vivo physical-state

transitions relating to disease causing processes. Such compounds, compositions, or conditions can be exploited to treat (e.g., reverse) or prevent the disease itself, the cause of the disease, or the symptoms of the disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:185606 USPATFULL

TITLE: Rapid identification of conditions, compounds, or compositions that inhibit, prevent, induce, modify, or reverse transitions of physical state

INVENTOR(S): Levinson, Douglas, Sherborn, MA, UNITED STATES

| | NUMBER | KIND | DATE |
|---------------------|----------------|------|--------------|
| PATENT INFORMATION: | US 2002098518 | A1 | 20020725 |
| APPLICATION INFO.: | US 2001-994585 | A1 | 20011127 (9) |

| | NUMBER | DATE |
|-----------------------|--|---------------|
| PRIORITY INFORMATION: | US 2000-253629P | 20001128 (60) |
| DOCUMENT TYPE: | Utility | |
| FILE SEGMENT: | APPLICATION | |
| LEGAL REPRESENTATIVE: | PENNIE AND EDMONDS, 1155 AVENUE OF THE AMERICAS, NEW YORK, NY, 100362711 | |
| NUMBER OF CLAIMS: | 80 | |
| EXEMPLARY CLAIM: | 1 | |
| LINE COUNT: | 3080 | |

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d his

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FILE 'MEDLINE, USPATFULL, DGENE, FSTA, BIOSIS, WPIDS, EMBASE, BIOTECHDS'
ENTERED AT 16:19:15 ON 21 JAN 2005

L1 7830 S HYPERCALCIURIA
L2 1061 S L1 AND RISK
L3 342 S HYPERCALCIURIA AND (INCREASE RISK OR PREDISPOSITION OR RISK F
L4 1 S L2 AND INCREASE RISK

=> s l3 and osteoporosis
L5 66 L3 AND OSTEOPOROSIS

=> s l5 and postmenopause
L6 15 L5 AND POSTMENOPAUSE

=> d l6 ti abs ibib tot

L6 ANSWER 1 OF 15 MEDLINE on STN
TI Review of risk factors for **osteoporosis** with particular reference to a possible aetiological role of dietary salt.
AB Laboratory animal, clinical and epidemiological studies in the published literature have been reviewed in order to establish whether excessive salt intake is an important **risk factor** for the development of **osteoporosis** and whether an intervention strategy based on salt restriction would be beneficial in the prevention of **osteoporosis**. Genetic factors appear to be far more important than the combination of nutritional, hormonal, environmental and lifestyle factors in the pathogenesis of **osteoporosis**. The most important single non-genetic factor is oestrogen deficiency in postmenopausal women. Preventive measures should be aimed at maximizing peak bone mass at skeletal maturity and retarding bone loss thereafter. Apart from postmenopausal oestrogen deficiency, various factors have been

incriminated as risk factors for **osteoporosis**, and these include age at menarche, age at and years since menopause, insufficient physical exercise, alcohol, smoking, low calcium intake, low or high protein intake and high intake of phosphorus, sodium or caffeine. Many of the risk factors are considered to be weak, although when combined they could impact significantly on bone health. Increased intakes of various nutritional factors (potassium, magnesium, zinc, vitamin C), fibre and alkaline-producing fruit and vegetables favour adult bone health. Calcium homeostasis is normally well regulated such that increased calcium loss via the urine leads to increased calcium absorption from the gut. However, the duration of this adaptive process may be greater than that of many of the studies demonstrating that increased salt intake leads to both increased sodium and calcium in the urine. In any case, higher urinary calcium output appears to be seen only in a minority of humans in response to increased salt intake. As numerous factors-genetic, nutritional, hormonal and lifestyle-are involved in the maintenance of calcium homeostasis, it is difficult to devise human studies which adequately take into account all the important factors. Another difficulty is that many past studies have relied on imprecise methods for the measurement of bone resorption. Nor have studies based on the use of the laboratory rat produced clear answers to the problem because the rat, as a species, is uniquely deficient in its ability to handle the relevant minerals. Limited studies to date indicate that increased sodium intake neither exerts a consistent effect on various biomarkers of bone health nor leads to irreversible changes in the bone modelling process in men or in pre- or postmenopausal women. We conclude from the available evidence that increased sodium (or salt) intake is not an important **risk factor** for **osteoporosis** and that a reduction of salt intake from 9 to 6g/day in the diet would not be beneficial as an intervention measure in the prevention of **osteoporosis**. More research is needed to (i) assess the effects (especially long-term) of various nutrients including sodium on bone health, (ii) assess the long-term value of any intervention strategy involving reduced intake of a particular nutrient such as sodium; and (iii) determine whether subpopulations exist particularly in the elderly (e.g. sodium-responsive subjects) in which adaptation to sodium-induced **hypercalciuria** may be compromised. General prudence dictates that excessively high levels of dietary salt should be eschewed by those persons with raised blood pressure or a limited range of genetic disorders. However, for the generally healthy person there is no sound evidence that the consumption of salt at the present average level of 9g/day constitutes a **risk factor** for **osteoporosis**.

ACCESSION NUMBER: 2000183656 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10717363
TITLE: Review of risk factors for **osteoporosis** with particular reference to a possible aetiological role of dietary salt.
AUTHOR: Cohen A J; Roe F J
CORPORATE SOURCE: Toxicology Advisory Services, Hamilton House, 17 Cedar Road, Sutton, Surrey, SM2 5DA, UK.
SOURCE: Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association, (2000 Feb-Mar) 38 (2-3) 237-53. Ref: 123
Journal code: 8207483. ISSN: 0278-6915.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200005
ENTRY DATE: Entered STN: 20000518
Last Updated on STN: 20000518
Entered Medline: 20000509

L6 ANSWER 2 OF 15 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
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TI Subchondral fractures of the femoral head: A review of seven cases.

AB Objective. - To describe the main characteristics of subchondral fractures of the femoral head. Case-Reports. - The seven patients, five women and two men, with a mean age of 50 years (37-76 years), presented with mechanical pain in the groin. Bone loss was the main **risk factor**. Two patients had postmenopausal **osteoporosis** (including one with a history of ovariectomy at 30 years of age), two had **osteoporosis** induced by glucocorticoid therapy given after transplantation (liver and allogeneic bone marrow, respectively), one had an ACTH-producing adenoma, and one had femoral osteopenia at a site of topical glucocorticoid therapy for atopic dermatitis. The remaining patient had osteopenia and a history of smoking. Phosphate and calcium levels were normal in five patients. One patient had isolated hypocalciuria and another had moderate proximal tubular disease with phosphate wasting and **hypercalciuria**. Magnetic resonance imaging (MRI) disclosed a subcapital line of low signal on T1- and T2-weighted sequences surrounded by an area of variable size generating low signal on T1 images and high signal on T2 images, with postgadolinium enhancement, denoting marrow edema. Complete elimination of weight bearing for 6 weeks, symptomatic agents, and treatment of the underlying causes of bone insufficiency were used in all seven patients. Mean follow-up was 2.4 years (range, 11-39 years). No cases of osteonecrosis were recorded. Conclusion. - Several cases of subchondral fracture have been reported in the literature. Bone insufficiency was the main **risk factor** in all the patients. .COPYRG. 2004 Elsevier SAS. All rights reserved.

ACCESSION NUMBER: 2004145602 EMBASE

TITLE: Subchondral fractures of the femoral head: A review of seven cases.

AUTHOR: Gerot I.L.; Demondion X.; Louville A.B.; Delcambre B.; Cortet B.

CORPORATE SOURCE: I.L. Gerot, Rheumatology Department, Lille Teaching Hospital, 59037 Lille, France. igerot@chru-lille.fr

SOURCE: Joint Bone Spine, (2004) 71/2 (131-135).

Refs: 19

ISSN: 1297-319X CODEN: JBSPFA

PUBLISHER IDENT.: S 1297-319X(03)00050-2

COUNTRY: France

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 005 General Pathology and Pathological Anatomy

033 Orthopedic Surgery

037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

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TI Pharmacotherapeutics for **osteoporosis** prevention and treatment.

AB **Osteoporosis** is a silent disease that affects 10 million Americans; 80% of those affected are women. Although the disease is more common in postmenopausal Caucasian women, all ages and races are at risk. **Osteoporosis** can be a debilitating disease that can cause pain, fractures, depression, and social withdrawal. Signs of **osteoporosis** include kyphosis, loss of height, and protrusion of the abdomen. Because symptoms generally do not occur until after the disease has progressed, clinicians should include **osteoporosis** screening and preventative education as part of the regular gynecologic care. Diagnosis is typically made by a dual energy x-ray absorptiometry (DEXA) scan. Treatment consists of dietary and lifestyle changes, along with pharmacologic intervention. Although hormone therapy has been shown to be effective in preventing **osteoporosis**, the risks of

long-term treatment with HRT are discussed. The following effective treatment options for women who have been diagnosed with the disease are discussed: bisphosphonates, calcitonin, and selective estrogen receptor modulators (SERMs). Because midwives regularly care for women of all ages, they are ideal candidates to provide women with preventative education, screening, counseling, and treatment. .COPYRGT. 2003 American College of Nurse-Midwives.

ACCESSION NUMBER: 2004141618 EMBASE
TITLE: Pharmacotherapeutics for **osteoporosis** prevention and treatment.
AUTHOR: Davidson M.R.
CORPORATE SOURCE: Dr. M.R. Davidson, 44108 Bristow Circle, Ashburn, VA 20147, United States
SOURCE: Journal of Midwifery and Women's Health, (2003) 48/1 (39-52).
Refs: 49
ISSN: 1526-9523 CODEN: JMWHAA
PUBLISHER IDENT.: S 1526-9523(02)00359-8
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 010 Obstetrics and Gynecology
017 Public Health, Social Medicine and Epidemiology
033 Orthopedic Surgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English

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TI Prevention and treatment of corticosteroid-induced **osteoporosis** in the elderly.

AB Corticosteroid-induced **osteoporosis** is a serious complication of corticosteroid therapy. Bone loss occurs within the first three to six months of corticosteroid use. Fracture rates can be quite high within the first year of corticosteroid therapy. All patients on or starting corticosteroids need to have assessments for secondary causes of **osteoporosis**, counseling for modification of risk factors, and measures taken to prevent fractures. All patients should have a calcium intake of 1200-1500 mg per day and vitamin D supplementation of 800-1000 IU per day. Bisphosphonates are currently the most potent antiresorptive agent. This class of medication has also been shown to reduce the occurrence of vertebral fractures. Bisphosphonate therapy is the recommended first-line agent both for the prevention and treatment of corticosteroid-induced **osteoporosis**.

ACCESSION NUMBER: 2003177353 EMBASE
TITLE: Prevention and treatment of corticosteroid-induced **osteoporosis** in the elderly.
AUTHOR: Boulos P.; Papaioannou A.; Adachi J.D.
CORPORATE SOURCE: Dr. J.D. Adachi, 501-25 Charlton Ave E, Hamilton, Ont. L8N 1Y2, Canada. jd.adachi@sympatico.ca
SOURCE: Annals of Long-Term Care, (1 Jan 2003) 11/1 (42-48).
Refs: 51
ISSN: 1524-7929 CODEN: ALTCHF
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 003 Endocrinology
020 Gerontology and Geriatrics
033 Orthopedic Surgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English

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TI Clinical evaluation for **osteoporosis**.

AB The clinical evaluation of the osteoporotic patient should include a careful assessment of risk factors for low bone mass, falls, and fractures; quantitation of BMD; a thorough medical history and physical examination; and a targeted set of laboratory, radiographic, and other diagnostic studies as indicated. Among the elderly, vitamin D deficiency ranks high as one of the most underdiagnosed and yet reversible causes of **osteoporosis**. Regardless of age, every patient with low bone mass or fractures deserves an evaluation to uncover reversible, treatable disorders and to detect serious underlying illnesses.

ACCESSION NUMBER: 2003174526 EMBASE

TITLE: Clinical evaluation for **osteoporosis**.

AUTHOR: Becker C.

CORPORATE SOURCE: Dr. C. Becker, Toni Stabile Osteoporosis Center, Columbia Presbyterian Medical Center, Harkness Pavilion-904, 180 Fort Washington Avenue, New York, NY 10032, United States. cb2006@columbia.edu

SOURCE: Clinics in Geriatric Medicine, (2003) 19/2 (299-320).

Refs: 137

ISSN: 0749-0690 CODEN: CGMEE6

PUBLISHER IDENT.: S 0749-0690(02)00068-X

COUNTRY: United States

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 003 Endocrinology
017 Public Health, Social Medicine and Epidemiology
020 Gerontology and Geriatrics
037 Drug Literature Index
038 Adverse Reactions Titles

LANGUAGE: English

SUMMARY LANGUAGE: English

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TI **Osteoporosis** - An overview.

AB **Osteoporosis** is a systemic progressive disease with important clinical complications because of the fractures that arise and cause morbidity in especially the aging postmenopausal women. It is recognized as an important public health problem because of the significant morbidity and mortality associated with its complication, namely fractures of the proximal femur (hip), vertebrae (spine), distal forearm, proximal humerus, pelvis, and other skeletal sites. Compared with other osteoporotic fractures, however, fractures of the hip incur the greatest morbidity and direct medical costs for health services. There are now a variety of treatments available for the management of **osteoporosis**. The inhibitors of bone resorption, which include calcium, the vitamin Ds, bisphosphonates, calcitonins and gonadal steroids have been variously shown to prevent bone loss or to reduce fractures. On the other hand bone formation stimulating agents as fluorides must be considered also. However, prevention of **osteoporosis** during the teen and early adult years is far superior to any of the treatment for older individuals with identification of risk factors, careful examination and a few simple diagnostic tests. The purpose of this review is to provide an general overview of an **osteoporosis**.

ACCESSION NUMBER: 2002180824 EMBASE

TITLE: **Osteoporosis** - An overview.

AUTHOR: Iqbal M.M.; Mahmud S.Z.

CORPORATE SOURCE: M.M. Iqbal, Department of Epidemiology, School of Public Health, University of Alabama at Birmingham, Birmingham, AL, United States

SOURCE: Bangladesh Journal of Obstetrics and Gynecology, (2001) 16/1 (27-34).

Refs: 20

ISSN: 1018-4287 CODEN: BJOGFX
 COUNTRY: Bangladesh
 DOCUMENT TYPE: Journal; General Review
 FILE SEGMENT: 010 Obstetrics and Gynecology
 017 Public Health, Social Medicine and Epidemiology
 033 Orthopedic Surgery
 037 Drug Literature Index
 038 Adverse Reactions Titles
 LANGUAGE: English
 SUMMARY LANGUAGE: English

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TI Complementary therapies for reducing the risk of **osteoporosis** in patients receiving luteinizing hormone-releasing hormone treatment/orchiectomy for prostate cancer: A review and assessment of the need for more research.

AB **Osteoporosis** in women has received a substantial amount of attention, but its impact in men is also significant and noteworthy. Those men who benefit from treatment for prostate cancer with androgen deprivation therapy (ADT) may also be at a higher risk for **osteoporosis**. Pharmacologic approaches to reduce this risk have received some attention. For example, agents such as bisphosphonates, estrogen receptor-binding drugs (diethylstilbestrol, tamoxifen, and raloxifene), calcitonin, and fluoride are some of the more promising interventions that have been previously outlined. In addition, statin drugs, or hepatic 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors, have recently been hypothesized to lower **osteoporosis** risk. However, complementary therapies, which may also have an impact on reducing **osteoporosis** risk, have not received attention. Dietary and supplemental calcium and vitamin D have been shown, in some preliminary investigations, to maintain bone density in women and men. Numerous healthy and affordable dietary sources of this mineral and vitamin exist, and large intakes can be realistically achieved through proper education. Similarly, the supplemental dosages required to impact risk have been moderate, appear to be safe, are of low cost, and thus may provide an additional route for reducing risk, especially if these interventions are initiated at the start of medical treatment. More studies in men receiving ADT are needed because the existing work has mostly focused on men without castrate levels of male hormone. Additionally, many studies with conventional and nonconventional agents have only focused on individuals with baseline **osteoporosis**, rather than normal bone mineral densities or osteopenia. Other promising complementary therapies, such as weight-bearing exercise and abstaining from smoking, may also be of benefit. Newer estrogenic-type supplements (eg, ipriflavone) appear interesting and have some preliminary data, but more research is desperately required to determine their actual impact and potential for adverse effects (such as lymphocytopenia from a recent trial). Simple, inexpensive, and potentially effective dietary and supplemental approaches to reduce the risk of **osteoporosis** in men exist, and they should be discussed with patients. Whether these approaches effectively reduce the risk of **osteoporosis** in men receiving androgen ablation remains to be determined. The possibility is intriguing, and future research is needed. In the meantime, it is important to keep in mind that these complementary approaches are, at the very least, an integral part of the conventional options used today to the reduce the risk of **osteoporosis** in men and women. .COPYRGT. 2002 Elsevier Science Inc.

ACCESSION NUMBER: 2002131130 EMBASE

TITLE: Complementary therapies for reducing the risk of **osteoporosis** in patients receiving luteinizing hormone-releasing hormone treatment/orchiectomy for prostate cancer: A review and assessment of the need for more research.

AUTHOR: Moyad M.A.
CORPORATE SOURCE: M.A. Moyad, Department of Urology, University of Michigan
Med. Center, 1500 East Medical Center Drive, Ann Arbor, MI
48109-0330, United States. moyad@umich.edu
SOURCE: Urology, (2002) 59/4 SUPPL. 1 (34-40).
Refs: 58
ISSN: 0090-4295 CODEN: URGYAZ
PUBLISHER IDENT.: S 0090-4295(01)01174-8
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 003 Endocrinology
016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English

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TI American association of clinical endocrinologists 2001 medical guidelines
for clinical practice for the prevention and management of postmenopausal
osteoporosis.

ACCESSION NUMBER: 2001272656 EMBASE
TITLE: American association of clinical endocrinologists 2001
medical guidelines for clinical practice for the prevention
and management of postmenopausal **osteoporosis**.

SOURCE: Endocrine Practice, (2001) 7/4 (294-312).
Refs: 87
ISSN: 1530-891X CODEN: EPNRAT

COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
010 Obstetrics and Gynecology
014 Radiology
033 Orthopedic Surgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English

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TI **Osteoporosis** in women with spinal cord injuries.

AB Decreased bone density and increased fracture risk are seen in patients
with SCI. The bone resorption rate is markedly increased.
Hypercalciuria, low PTH, and low 1,25 (OH)(2) vitamin D are
commonly seen. Bed-rest studies show similar findings, but of lesser
magnitude. Therapies to treat or prevent **osteoporosis** include
optimal nutrition (with care to avoid exacerbating **hypercalciuria**
) . Weight-bearing or functional electrical stimulation cycle ergometry may
prevent some of the bone loss, especially in acutely injured patients.
Estrogen should be considered in postmenopausal or amenorrheic women, but
not if they are at high risk of thromboembolism. More research on effects
of estrogen is needed in this population. Bisphosphonates may also help
prevent the acute bone loss; oral routes must not be used in recumbent
patients. Thiazides could be useful as adjunct therapy.

ACCESSION NUMBER: 2001031537 EMBASE
TITLE: **Osteoporosis** in women with spinal cord injuries.
AUTHOR: Ott S.M.
CORPORATE SOURCE: Dr. S.M. Ott, Division of Metabolism, University of
Washington, 1959 NE Pacific Street, Seattle, WA 98195-6426,
United States
SOURCE: Physical Medicine and Rehabilitation Clinics of North
America, (2001) 12/1 (111-131).

Refs: 64
ISSN: 1047-9651 CODEN: PMRAFZ
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 019 Rehabilitation and Physical Medicine
031 Arthritis and Rheumatism
033 Orthopedic Surgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English

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TI Risk of calcium oxalate nephrolithiasis after calcium or combined calcium
and calcitriol supplementation in postmenopausal women.

AB Although calcium supplementation can cause **hypercalciuria**, the
risk of nephrolithiasis has been shown to decrease rather than increase
among subjects who had a higher calcium intake. **Hypercalciuria**
is also a well-established side effect of calcitriol administration.
However, the risk of nephrolithiasis is not well defined. The present
study was undertaken to prospectively determine the effect of calcium with
or without calcitriol on physicochemical risk factors associated with
calcium oxalate nephrolithiasis in Thai postmenopausal women with
osteoporosis. Subjects consisted of 53 Thai women more than 10
years postmenopausal who were randomly allocated to receive 750 mg of
calcium carbonate supplement alone (n = 28) or 750 mg of calcium carbonate
plus 0.5 µg calcitriol (n = 25) daily. Mean ± SEM for age was 65.3
± 1.1 years, body weight 53.5 ± 1.3 kg. Urine samples for
biochemical assays were collected at baseline and 3 months after
treatment. Supersaturation for calcium oxalate stone formation was
assessed from the 24 h urine constituents by the Tiselius's index,
AP(CaOx). Three months of calcium supplement alone resulted in a modest,
but not significant, increase in urinary calcium (baseline, 2.90 ± 0.43
mmol/day; after treatment 3.58 ± 0.54 mmol/day) with no change in
urinary oxalate, citrate or magnesium. In contrast, calcium together with
calcitriol caused a significant increase in urinary calcium (baseline,
2.87 ± 0.41 mmol/day; after treatment, 4.08 ± 0.57 mmol/day; p <
0.05). No significant change in other urine constituents after treatment
with calcium and calcitriol was detected. Therefore, AP(CaOx) did not
significantly increase either after calcium alone (baseline, 1.17 ±
0.39; after treatment, 1.36 ± 0.28) or after calcium plus calcitriol
(baseline, 1.09 ± 0.17; after treatment, 1.09 ± 0.19). However,
after treatments, 12 subjects (23%) - 6 receiving calcium supplement alone
and 6 receiving calcium plus calcitriol supplement - had high AP(CaOx)
values (greater than the upper limit of 95% CI for AP(CaOx) derived from
non-stone-forming Thai women). The post-treatment/baseline ratio was 3.21
± 0.74 for urinary calcium, 1.01 ± 0.19 for urinary oxalate, and
2.23 ± 0.42 (median 1.15) for AP(CaOx). The post-treatment/baseline
ratio of calcium, but not for urinary oxalate, had a significant
correlation with the post-treatment/baseline ratio of AP(CaOx). Our
findings suggest that the alteration in the risk of calcium oxalate
nephrolithiasis based on urinary composition is related to the alteration
in urinary calcium. The risk of calcium oxalate nephrolithiasis does not
increase significantly after calcium or combined calcium and calcitriol
supplement in the majority of postmenopausal women with
osteoporosis.

ACCESSION NUMBER: 2000318421 EMBASE

TITLE: Risk of calcium oxalate nephrolithiasis after calcium or
combined calcium and calcitriol supplementation in
postmenopausal women.

AUTHOR: Domrongkitchaiporn S.; Ongphiphadhanakul B.; Stichtantrakul
W.; Piaseu N.; Chansirikarn S.; Puavilai G.; Rajatanavin R.

CORPORATE SOURCE: Dr. S. Domrongkitchaiporn, Department of Medicine,

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SOURCE: Osteoporosis International, (2000) 11/6 (486-492).

Refs: 37

ISSN: 0937-941X CODEN: OSINEP

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 028 Urology and Nephrology

033 Orthopedic Surgery

037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

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on STN

TI Review of risk factors for **osteoporosis** with particular
reference to a possible aetiological role of dietary salt.

AB Laboratory animal, clinical and epidemiological studies in the published
literature have been reviewed in order to establish whether excessive salt
intake is an important **risk factor** for the development
of **osteoporosis** and whether an intervention strategy based on
salt restriction would be beneficial in the prevention of
osteoporosis. Genetic factors appear to be far more important than
the combination of nutritional, hormonal, environmental and lifestyle
factors in the pathogenesis of **osteoporosis**. The most important
single non-genetic factor is oestrogen deficiency in postmenopausal women.
Preventive measures should be aimed at maximizing peak bone mass at
skeletal maturity and retarding bone loss thereafter. Apart from
postmenopausal oestrogen deficiency, various factors have been
incriminated as risk factors for **osteoporosis**, and these include
age at menarche, age at and years since menopause, insufficient physical
exercise, alcohol, smoking, low calcium intake, low or high protein intake
and high intake of phosphorus, sodium or caffeine. Many of the risk
factors are considered to be weak, although when combined they could
impact significantly on bone health. Increased intakes of various
nutritional factors (potassium, magnesium, zinc, vitamin C), fibre and
alkaline-producing fruit and vegetables favour adult bone health. Calcium
homeostasis is normally well regulated such that increased calcium loss
via the urine leads to increased calcium absorption from the gut. However,
the duration of this adaptive process may be greater than that of many of
the studies demonstrating that increased salt intake leads to both
increased sodium and calcium in the urine. In any case, higher urinary
calcium output appears to be seen only in a minority of humans in response
to increased salt intake. As numerous factors - genetic, nutritional,
hormonal and lifestyle - are involved in the maintenance of calcium
homeostasis, it is difficult to devise human studies which adequately take
into account all the important factors. Another difficulty is that many
past studies have relied on imprecise methods for the measurement of bone
resorption. Nor have studies based on the use of the laboratory rat
produced clear answers to the problem because the rat, as a species, is
uniquely deficient in its ability to handle the relevant minerals. Limited
studies to date indicate that increased sodium intake neither exerts a
consistent effect on various biomarkers of bone health nor leads to
irreversible changes in the bone modelling process in men or in pre- or
postmenopausal women. We conclude from the available evidence that
increased sodium (or salt) intake is not an important **risk**
factor for **osteoporosis** and that a reduction of salt
intake from 9 to 6 g/day in the diet would not be beneficial as an
intervention measure in the prevention of **osteoporosis**. More
research is needed to (i) assess the effects (especially long-term) of
various nutrients including sodium on bone health, (ii) assess the
long-term value of any intervention strategy involving reduced intake of a
particular nutrient such as sodium; and (iii) determine whether
subpopulations exist particularly in the elderly (e.g. sodium-responsive

subjects) in which adaptation to sodium-induced **hypercalciuria** may be compromised. General prudence dictates that excessively high levels of dietary salt should be eschewed by those persons with raised blood pressure or a limited range of genetic disorders. However, for the generally healthy person there is no sound evidence that the consumption of salt at the present average level of 9 g/day constitutes a **risk factor** for **osteoporosis**. (C) 2000 Elsevier Science Ltd.

ACCESSION NUMBER: 2000091904 EMBASE
TITLE: Review of risk factors for **osteoporosis** with particular reference to a possible aetiological role of dietary salt.
AUTHOR: Cohen A.J.; Roe F.J.C.
CORPORATE SOURCE: A.J. Cohen, Toxicology Advisory Services, Hamilton House, 17 Cedar Road, Sutton, Surrey SM2 5AD, United Kingdom
SOURCE: Food and Chemical Toxicology, (2000) 38/2-3 (237-253).
Refs: 123
ISSN: 0278-6915 CODEN: FCTOD7
PUBLISHER IDENT.: S 0278-6915(99)00145-3
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

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TI [**Osteoporosis**].
OSTEOPOROSE.

ACCESSION NUMBER: 2000027931 EMBASE
TITLE: [**Osteoporosis**].
OSTEOPOROSE.
AUTHOR: Rohart C.; Benhamou C.L.
CORPORATE SOURCE: Dr. C. Rohart, Service de Rhumatologie, Hopital Porte Madeleine, CHR Orleans, 45032 Orleans Cedex I, France
SOURCE: Revue du Praticien, (1 Jan 2000) 50/1 (85-92).
ISSN: 0035-2640 CODEN: REPRA3
COUNTRY: France
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 006 Internal Medicine
LANGUAGE: French

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TI **Osteoporosis** and systemic lupus erythematosus: Etiology and treatment strategies.

AB The importance of bone loss in patients with systemic lupus erythematosus can not be over emphasized. Risk factors for **osteoporosis** in these patients include not only those that apply to the general population, but also many that are related to the underlying disease process, or its treatment. Ongoing research should attempt to clarify the prevalence of low bone mass in lupus, as well as the pathogenic mechanisms applying especially to this population. Strategies for the prevention and treatment of bone loss in lupus patients do not differ significantly from those in the general population. Special attention must be given to the prevention of steroid-induced bone loss, as well as to the gonadal effects of cytotoxic agents.

ACCESSION NUMBER: 96283369 EMBASE
DOCUMENT NUMBER: 1996283369
TITLE: **Osteoporosis** and systemic lupus erythematosus: Etiology and treatment strategies.
AUTHOR: Segal L.G.; Lane N.E.
CORPORATE SOURCE: Division of Rheumatology, Box 0868, University of California, San Francisco, CA 94143, United States
SOURCE: Annales de Medecine Interne, (1996) 147/4 (281-289).

ISSN: 0003-410X CODEN: AMDIBO
COUNTRY: France
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 006 Internal Medicine
031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English; French

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TI Bone and the 'Comforts of Life'.

AB Coffee drinking, smoking and especially alcohol abuse are considered to be risk factors for fractures and **osteoporosis**. Caffeine causes acute increase in urinary calcium excretion, but epidemiological evidence for the effects of coffee consumption on the risk of fractures is contradictory. Many, (but not all) studies point to decreased bone mass or increased fracture risk in smokers. Alcohol abuse is associated with deleterious changes in bone structure detected by histomorphometry, and with a decrease in bone mineral density (BMD). These changes may also be produced by factors commonly associated with alcohol abuse, e.g. nutritional deficiencies, liver damage and hypogonadism. Alcohol, however, has clear-cut direct effects on bone and mineral metabolism. Acute alcohol intoxication causes transitory hypoparathyroidism with resultant hypocalcaemia and **hypercalciuria**. As assessed by serum osteocalcin levels, prolonged moderate drinking decreases the function of osteoblasts, the bone-forming cells. In addition, chronic alcoholics are characterized by low serum levels of vitamin D metabolites. Thus, alcohol seems to have a direct toxic effect on bone and mineral metabolism. In contrast, it has recently been reported that moderate alcohol consumption by postmenopausal women may have a beneficial effect on bone.

ACCESSION NUMBER: 93250294 EMBASE

DOCUMENT NUMBER: 1993250294

TITLE: Bone and the 'Comforts of Life'.

AUTHOR: Laitinen K.; Valimaki M.

CORPORATE SOURCE: Research Unit of Alcohol Diseases, Heisinki University
Central Hospital, Tukholmankatu 8 F, SF-00290 Helsinki,
Finland

SOURCE: Annals of Medicine, (1993) 25/4 (413-425).

ISSN: 0785-3890 CODEN: ANMDEU

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 005 General Pathology and Pathological Anatomy
017 Public Health, Social Medicine and Epidemiology
033 Orthopedic Surgery

LANGUAGE: English

SUMMARY LANGUAGE: English

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TI [**Osteoporosis** due to hormone deficiency: Prevention by sex hormone substitution!].

OSTEOPOROSE DUE TO HORMONE DEFICIENCY: PREVENTION BY SEX HORMONE SUBSTITUTION!.

AB The postmenopausal withdrawal of estrogens results in a vicious circle, which is initiated by accelerated bone resorption, hypercalcemia, failure of the calcium retaining system ('escape' phenomenon), and hypercalcuria, and is associated with accelerated bone turnover, and a negative balance of calcium and bone metabolism. To maintain homeostasis, the waste of bone calcium has to be continually balanced by additional osteolysis. Bone resorption is inevitably followed by **osteoporosis** after a varying duration. Following intake of estrogens, the vicious circle is interrupted by deceleration of osteolysis and bone turnover, and a

positive balance of calcium and bone metabolism is restored.
Osteoporosis, as a consequence of postmenopausal endocrine deficiency, may therefore be prevented by a substitution with female sex steroids in due time.

ACCESSION NUMBER: 92068750 EMBASE
DOCUMENT NUMBER: 1992068750
TITLE: [Osteoporosis due to hormone deficiency:
Prevention by sex hormone substitution!].
OSTEOPOROSE DURCH ENDOKRINES DEFIZIT-SYNDROM. VERMEIDBAR
DURCH SEXUAL-HORMON-SUBSTITUTION!.

AUTHOR: Nocke W.
CORPORATE SOURCE: Abteilung fur Gynakologische Endokrinologie, Zentrum fur
Geburtshilfe und Frauenheilkunde, Rheinische
Friedrich-Wilhelms-Universitat, W-5300 Bonn-Venusberg,
Germany

SOURCE: Therapiewoche, (1992) 42/7 (350-357).
ISSN: 0040-5973 CODEN: THEWA6

COUNTRY: Germany
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 010 Obstetrics and Gynecology
033 Orthopedic Surgery
037 Drug Literature Index

LANGUAGE: German
SUMMARY LANGUAGE: German; English

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(FILE 'HOME' ENTERED AT 16:18:34 ON 21 JAN 2005)

FILE 'MEDLINE, USPATFULL, DGENE, FSTA, BIOSIS, WPIDS, EMBASE, BIOTECHDS'
ENTERED AT 16:19:15 ON 21 JAN 2005

L1 7830 S HYPERCALCIURIA
L2 1061 S L1 AND RISK
L3 342 S HYPERCALCIURIA AND (INCREASE RISK OR PREDISPOSITION OR RISK F
L4 1 S L2 AND INCREASE RISK
L5 66 S L3 AND OSTEOPOROSIS
L6 15 S L5 AND POSTMENOPAUSE

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E1 2 REED Y/AU
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E3 0 --> REED-GITOMER/AU
E4 1 REEDA A/AU
E5 1 REEDA C F/AU
E6 1 REEDAL D C/AU
E7 1 REEDAL D R/AU
E8 1 REEDAL DONNA R/AU
E9 3 REEDAL J S/AU
E10 1 REEDD J D/AU
E11 1 REEDDY K/AU
E12 1 REEDE COOLEY JR N/AU